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STRUCTURAL ALTERATIONS IN THE CORNEA FROM EXPOSURE TO INFRARED RADIATION

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This report summarizes our research on the interaction of infrared radiation, especially from high-intensity CO_2 TEA lasers, with the cornea. The research reported here was performed between April 1, 1986 and April 14, 1988. The report discusses threshold epithelial damage from single- and multiple-pulse exposures, material ejection from the anterior corneal surface, lesion histology, and possible damage mechanisms. Keywords: Laser effects; Laser hazards.

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Foreword

In conducting the research described in this report, the investigators adhered to the *Guide for the Care and Use of Laboratory Animals*, prepared by the Committee on Care and Use of Laboratory Animals of the Institute of Laboratory Animal Resources, National Research Council (DHEW Publication No. (NIH) 85-23, Revised Edition, 1985).

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Contents

Illustrations

Table

1.0 LASER

A Boston Laser Model 220S CO_2 TEA laser was used for all of the exposures in the research described below. The laser has a variable aperture which allows it to operate in the TEM_{00} mode which has a Gaussian profile. This was the only mode employed in our work. The pulse repetition frequency has a maximum of 20 Hertz for this laser. The beam irradiance profile was measured using a 64 element array of pyroelectric detectors made by Spiricon. A typical spatial profile of the beam is shown in Fig. 1. The 1/e radius of the beam is the off-axis distance at which the irradiance in the Gaussian profile is 1/e (36.8%) of its value at the center. For a Gaussian beam profile, the peak irradiance, I_0, is related to the total power, P, in the beam by $I_0 = P/A$, where A is the area within the 1/e radius. The nominal 1/e beam radius of the laser was 1.9 mm. The energy in each pulse was obtained with a Scientech power/energy meter. A Molectron pyroelectric detector was employed to determine the time profile of a pulse from which the pulsewidth was obtained. The pulsewidth was about 80 nanoseconds.

Fig. 1 Typical beam profile. Photograph of the output of the 64 element Spiricon pyroelectric detector. The center-to-center spacing of the elements is 0.2 mm. The 1/e radius of this beam is 1.86 mm.

2.0 ANIMALS

New Zealand white rabbits weighing 5 to 7 lb were used for the experiments. A 40:60 mixture of ketamine hydrochloride (100 mg/ml) and xylazine (20 mg/ml) was employed as a general anesthetic for rabbits and was excellent for stopping eye motion. In addition, proparacaine hydrochloride ophthalmic solution (Alcaine®) was applied topically to the cornea. The anesthetized animals were placed in a conventional holder and were positioned with the aid of a He-Ne alignment laser whose beam was colinear with the CO_2 laser output. The incident radiation was aligned perpendicular to the surface of the cornea. In all experiments, the cornea was irrigated about 20 s before exposure with a small amount of physiological saline at room temperature. At ~10 s before exposure, excess saline was blotted by holding an absorbent tissue against the limbus just below the area to be exposed. This process assured a reproducible "tear" layer. Because of the small amount of liquid involved, the 10-s interval between blotting and exposure was sufficient to ensure that the cornea's surface temperature returned to its steady state value. Following exposure, the eye was blinked manually to reform its natural tear film.

3.0 EPITHELIAL DAMAGE THRESHOLDS

We made measurements of four damage thresholds--one for a single 80 ns pulse and three for sequences of 80 ns pulses. For exposures at the single-pulse damage threshold we have observed and photographed material being ejected from the corneal surface. The ejection occurs with a delay of several microseconds after the laser pulse. Histology and slit-lamp observations reveal damage that appears different from that observed for longer duration exposures ($\gtrsim$ 1 ms). Certain features of this damage are consistent with mechanical or acoustic damage, but others are consistent with thermal damage. Temperature calculations reveal that the temperature gradient at the anterior tear surface is sufficient to drive an acoustic pressure wave; however, the temperature rise within the anterior epithelium is sufficiently high that a thermal damage mechanism cannot be ruled out. The remainder of this section includes an extensive discussion of material pertinent to our investigations of threshold damage from very short exposures to laser pulses of large irradiance.

During the two years of the grant, we have investigated threshold damage conditions for one single-pulse and three multiple-pulse corneal exposures, as listed in Table I. Also contained in Table I are data from Mueller and Ham (Ref. 1), who reported a damage threshold of 6 mJ/cm² for a 1.4 ns pulse and Zuclich et al (Ref. 2), who published threshold values of 660, 1080 and 360 mJ/cm² for exposures of 1.7, 25, and 250 ns, respectively. The values in the table for the data of Zuclich et al, which give the energy density on the beam axis, are twice what they reported; because, as they stated specifically, "Corneal radiant exposures were calculated by dividing the total incident energy by the area defined by the $1/e^2$ beam diameter, which was ~3 mm for all exposures." In contrast, the peak energy density is obtained by dividing the

TABLE I

N	PRF(Hz)	$d_{1/e}$(mm)	ED_{th}(mJ/cm²) (per pulse)	τ(ns)	ΔT_{max}(°C)+	$\frac{\Delta T}{\Delta Z}(\frac{°C}{cm})$§	Ref.
1	—	9.5*	6	1.4	1.8		1
1	—	2.12	660	1.7	65.0	1.4×10^5	2
1	—	2.12	1080	25	106.4	2.2×10^5	2
1	—	2.12	360	250	35.5	4.7×10^4	2
1	—	3.88	360	80	35.5	7.4×10^4	Our
2	1	3.72	300	80	30.5	5.3×10^4	Our
2	10	3.82	200	80	21.9	3.5×10^4	Our
8	10	3.80	228	80	32.0	4.0×10^4	Our

* Not a Gaussian beam. Ham and Mueller (Ref. 1) used an essentially flat energy density profile.

+ Maximum calculated temperature increase on the beam axis at 10 μm into the cornea.

§ Temperature gradient calculated between 0.1 and 0.2 μm into the cornea (anterior tearlayer).

incident energy by the area at the 1/e diameter, which explains the factor of two. It is important to note that Mueller and Ham's damage for the 6 mJ/cm² data point was observed 48 hours post-exposure, whereas our data and that of Zuclich et al had damage endpoints shortly after exposure.

Using Mueller and Ham's numbers (Ref. 1) we calculate a temperature increase of 1.8 °C on the beam axis 10 μm into the cornea. At a depth of 1 μm the temperature rise was ~2.5 °C. Thus, it is improbable that the damage they observed could be explained by a thermal model. For the data of Zuclich et al (Ref. 2), the corresponding temperature increases are 65.0, 106.4, and 35.5 °C at a depth of 10 μm and 136.4, 223.2, and 74.4 °C at a depth of 1 μm. For our single-pulse threshold we compute a temperature increase of 35.5 °C at a depth of 10 μm on the beam axis and 74.4 °C at a depth of 1 μm. Although the temperature increases for the Zuclich data are high, the energy absorbed in the anterior cornea is still well below the 2600 J/cm³ vaporization threshold for water even for the 25 nanosecond exposure.

Our multiple-pulse thresholds indicate peak temperature increases of 30.5, 21.9 and 32.0 °C at a depth of 10 μm on the beam axis. These preliminary multiple-pulse data need to be refined. It is apparent that the radiant exposure per pulse for the eight pulse-10 Hz exposure should not be greater than that for the two pulse-10 Hz exposure.

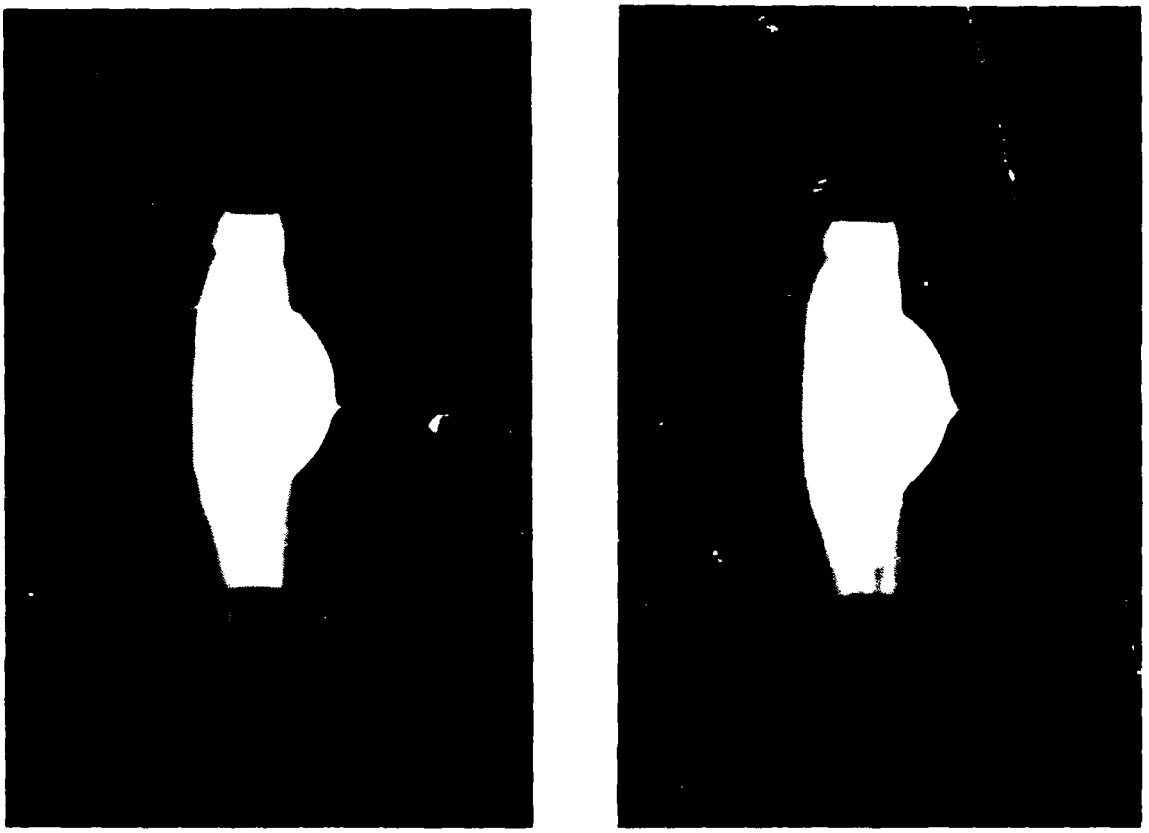
In their paper Zuclich et al (Ref. 2) firmly attribute damage to a thermal mechanism. This attribution is based on reasonable agreement between their data and the empirical thermal model due to Reed (Ref. 3). Our single-pulse data from Table I also is in good agreement with Reed's model. However, we note that the variability in the temperature rises calculated from Zuclich et al's data are inconsistent with thermal models based on a critical temperature (Refs.

4,5,6,). Moreover, the damage integral model has also led to inconclusive predictions for very short pulses (Ref. 7) and, indeed, has not been applied to Zuclich et al's data.

Other damage mechanisms cannot be rejected. For example, like Ham and Mueller (Ref. 1), Zuclich and Blankenstein reported hearing an "audible report from the cornea with lesion producing exposures" (Ref. 8). This suggests that one should at least consider possible acoustic damage mechanisms.

All of the damage thresholds in Table I are well below the vaporization threshold of water (2600 J/cm^3), thus ruling out the recoil momentum from rapid vaporization as the source of an acoustic pulse. However, the calculated temperature gradients at the anterior tear layer are all sufficient to produce a pressure gradient via a thermoelastic process (Ref. 9) that would exceed the 1 atm/cm cited by Mueller and Ham (Ref. 1) as being sufficient to damage cell membranes.

We have obtained additional evidence that is consistent with an acoustic mechanism for damage. Figure 2a shows material being ejected from the surface of the cornea of an enucleated eye at an exposure near the single-pulse damage threshold. At higher exposure levels (but still below the vaporization threshold), the plume becomes quite large--extending 10-20 mm from the corneal surface (cf. Fig. 2b). The mechanism for ejection of material is an intriguing mystery to be solved. It is perhaps due to rapid forward expansion of the tear surface, coupled with a reduction in the surface tension due to the temperature increase. Even more puzzling, however, is our preliminary finding that the ejection of material takes place several microseconds following the 80 ns laser pulse. The timing and photographic resolution must be refined before this phenomenon can be understood.



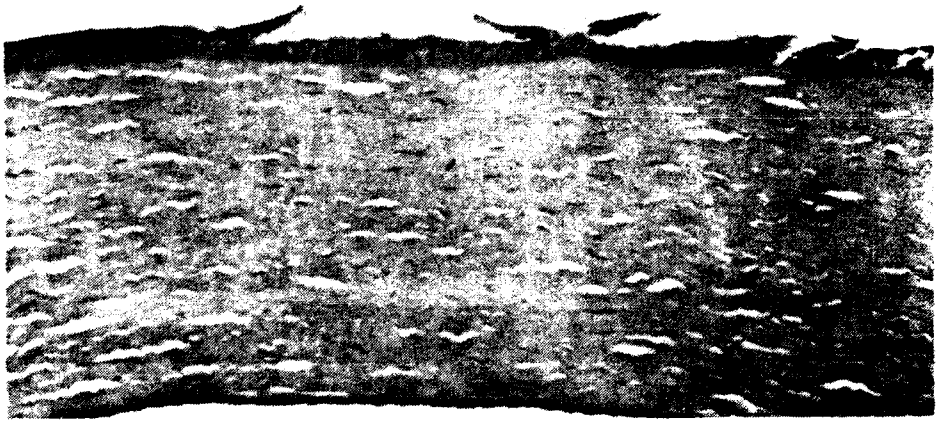
(a) (b)

Fig. 2 Effect of CO_2-TEA laser radiation on cornea. The 80 ns pulses were incident on an enucleated eye. In (a) the energy density was 460 mJ/cm^2 and in (b) it was 840 mJ/cm^2. These photographs are time exposures of the plume which is made visible by a HeNe laser beam that is colinear with the infrared beam.

Histology of near-threshold lesions shows features consistent with both acoustic and thermal damage. Figures 3 and 4 show a lesion resulting from a 397 mJ/cm^2 exposure, which is only about 10 percent above the damage threshold. The wing cells are lifted away from the surface in the center of the lesion, as might be expected for shock-wave induced damage. Moreover, the nuclei of these disrupted cells appear intact, with no apparent evidence of the coagulation that characterized threshold lesions for longer exposures where the damage was purely thermal (cf. Fig. 5).

At the higher magnification provided by transmission electron microscopy (TEM), however, nuclear damage in the superficial cells is evident. Figure 6 shows the epithelium at the center of a lesion resulting from an exposure of 405 mJ/cm^2. The damage is characterized by a degenerating superficial cell layer overlying a normal basal epithelium and stroma. The degenerating cells show a loss of well-defined organelles, along with an accumulation of amorphous, electron-dense material, and the development of vacuoles. At higher magnification, Fig. 7 clearly shows that the basal cells under the central damaged area are normal, as is the basement membrane and anterior stroma. There is a sharp demarcation between damaged and undamaged zones at the wound margin (cf. Fig. 8). Figures 9 and 10 are from the central wound area that resulted from an exposure of 755 mJ/cm^2, which is approximately two times the damage threshold. The essential nature of the damage is the same as for the lesion shown in Figs. 6-8, except that damage extends deeper into the epithelium. The basal cells remain intact, but their shape is distorted, perhaps due to their being pushed aside by the swelling evident in the overlying damaged area. Again, at higher magnification Fig. 10 shows that the basement membrane and anterior stroma are normal.

(a)



(b)

Fig. 3 Thick section of a cornea exposed by an 80 ns - 397 mJ/cm^2 pulse from a CO_2-TEA laser. In (a) the entire wound region is shown. The wound area was located with a 6 mm non-penetrating trephine cut - which is evident at the right margin of the photograph. The damage is indicated by the disrupted epithelial cells. In (b) the disrupted cells are shown at higher magnification. Damage is confined to the wing cells of the anterior epithelium (ref. Fig. 3).

(a)

(b)

Fig. 4 Higher magnification photographs of the damage region shown in Fig. 2. There is no apparent evidence of coagulation and the nuclei of the disrupted wing cells appear intact.

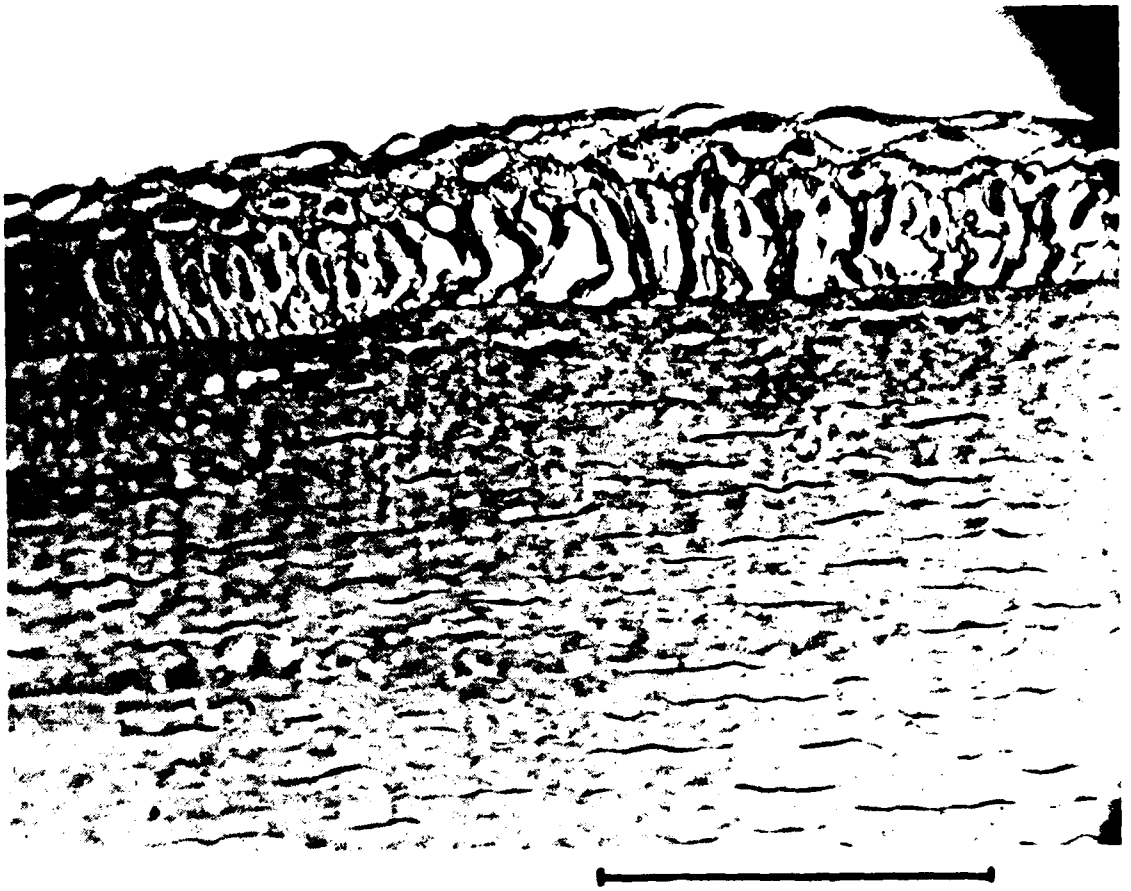


Fig. 5 Thick section of a cornea exposed at 11 W/cm² for 0.82 sec. This is slightly above the epithelial damage threshold. The animal was sacrificed one-half hour after the exposure. There is epithelial edema through all cell layers at the center of the exposed area. The disrupted area extends over about 0.6-0.7 mm and within it there is moderate cell disruption with edematous spaces between the cells. Outside this region (not shown) the cells are normal.

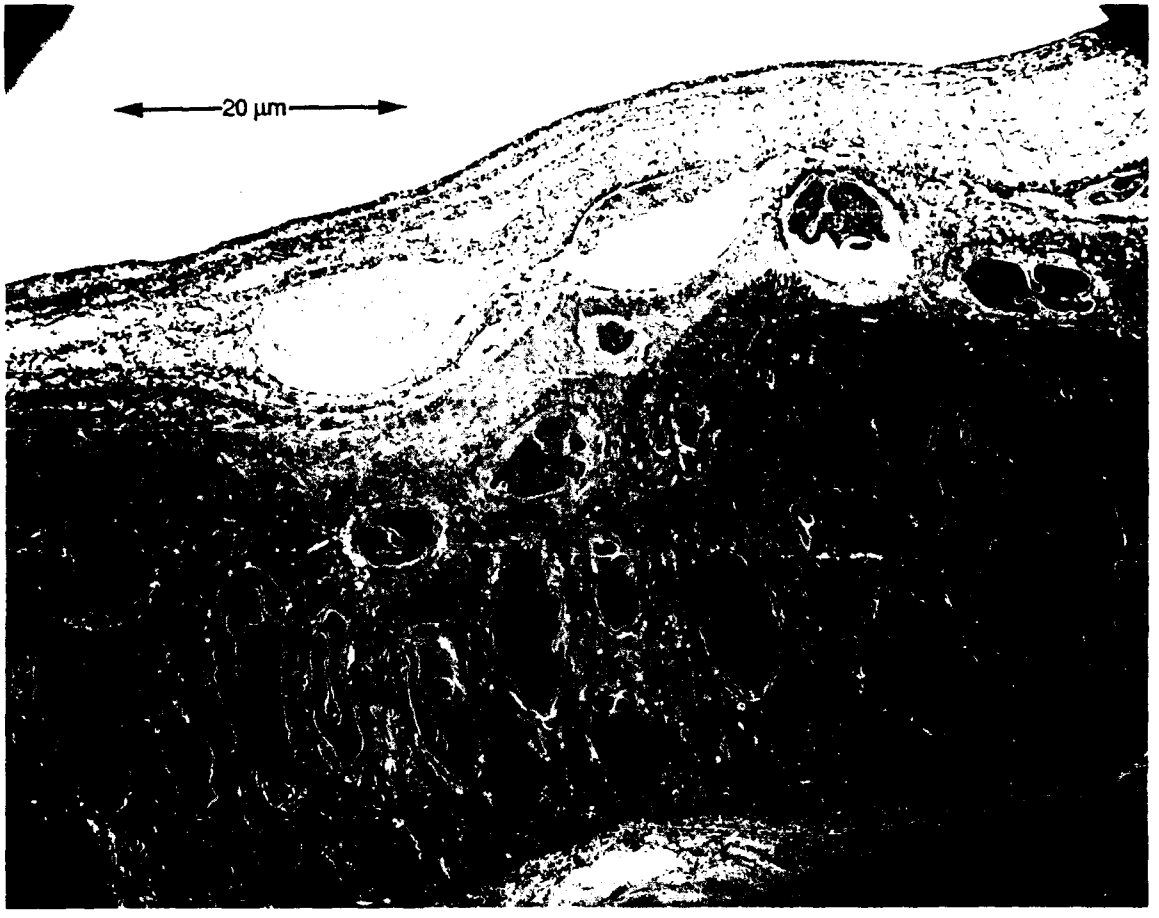


Fig. 6 Epithelium at the center of a lesion produced by an exposure of 405 mJ/cm², which is 1.12 times the damage threshold. Damage is characterized by a degenerating superficial cell layer that overlies normal basal cells. The degenerating cells show loss of well-defined organelles, accumulation of amorphous, electron dense material and vacuolation.

Fig. 7 Basal epithelium and anterior stroma at the center of the lesion shown in Fig. 6. These regions are completely normal, as is the basement membrane.

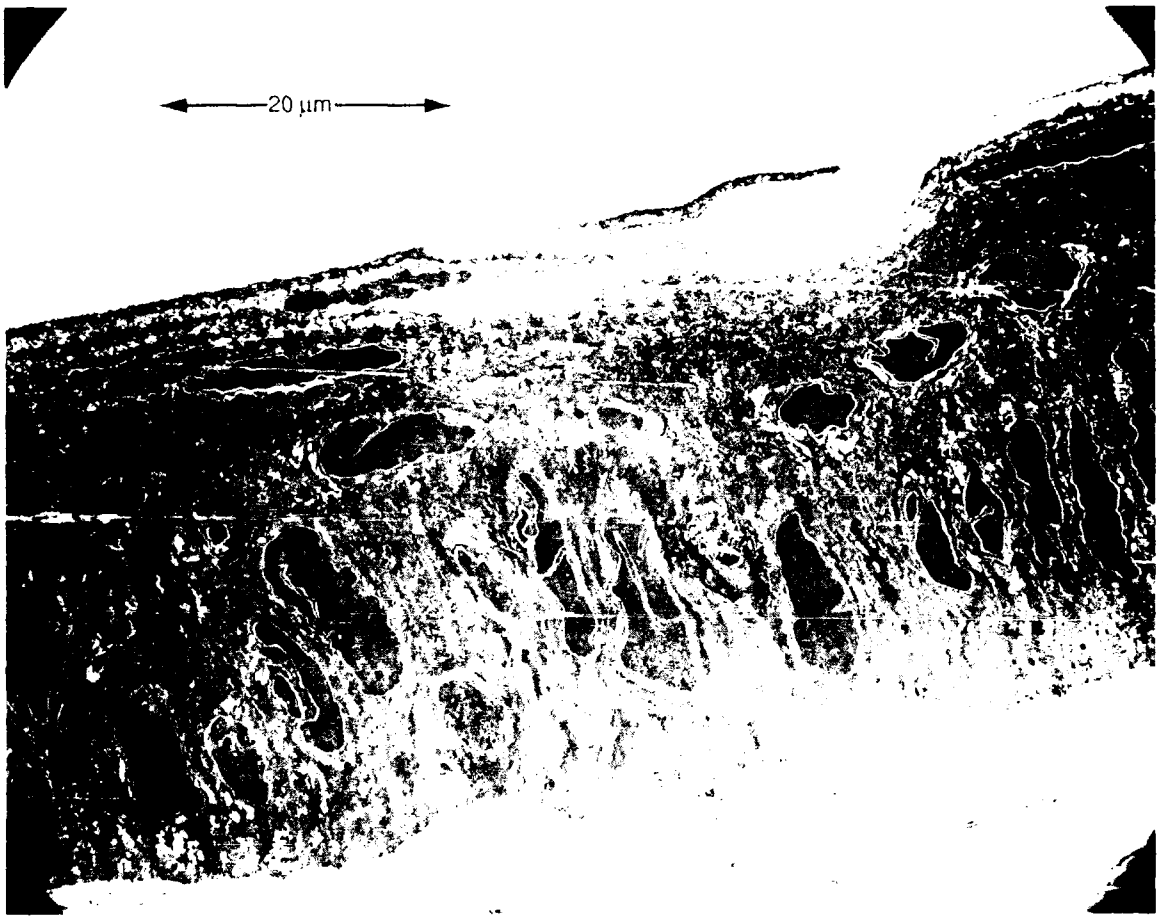


Fig. 8 Epithelium at the margin of the lesion shown in Figs. 6 and 7. The sharp demarcation between damaged and undamaged areas is evident.

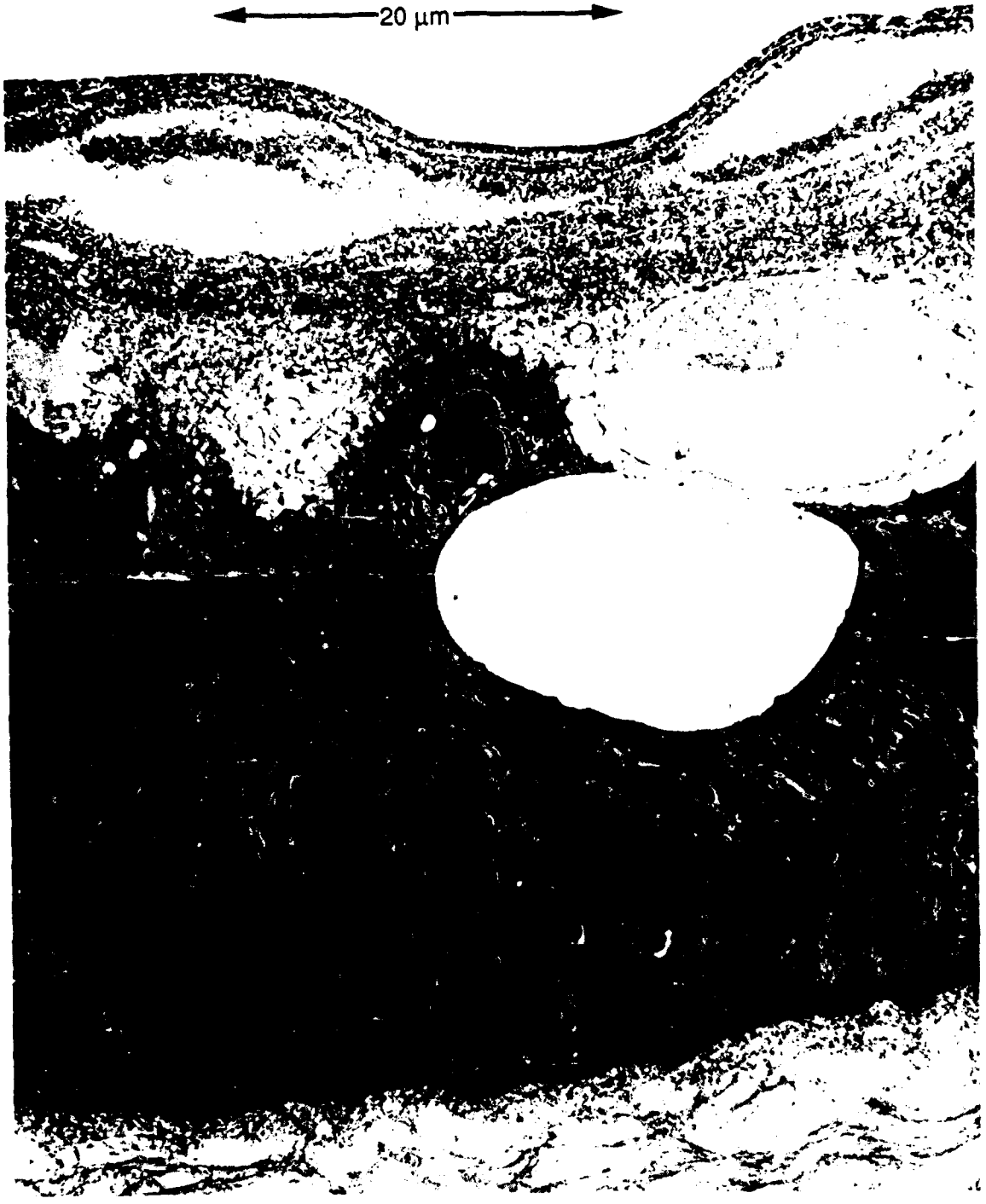


Fig. 9 Epithelium at the center of a lesion produced by an exposure of 755 mJ/cm², which is 2.1 times the damage threshold. The damage has the same characteristics as the near-threshold lesion shown in Fig. 6, except that it extends deeper into the epithelium. The basal cells remain intact, but their shape has been slightly distorted by the large vacuoles in the anterior epithelium.

Fig. 10 Basal epithelium and anterior stroma at the center of the lesion shown in Fig. 9. The distorted shape of the basal cells is evident, but the basement membrane and anterior stroma are normal.

Vacuolation, loss of well-defined organelles and the accumulation of electron dense material all are features noted in thermal lesions of the epithelium. Thus it is apparent that the damage may indeed contain a thermal component as well. Further research should be performed in order to clarify these observations and to combine them into a comprehensive model that can explain both the thermal and the mechanical features of the interaction.

4.0 DAMAGE MECHANISMS

a. Theoretical Acoustic Models

Several groups and individuals have published acoustic models for laser-generated acoustic waves in water. The model which seems most appropriate to our research is due to Sigrist and Kneubühl (Ref. 9), who studied laser-induced stress waves in liquids generated by the vaporization process and/or the thermoelastic effect. They improved upon a model for spherical pressure waves by Hu (Ref. 10). While neither of these models concern tissue damage, both contain results useful to a tissue damage model. The formulations include expressions for pressure as a function of position and time. However, Sigrist and Kneubühl's model is limited because it assumes instantaneous deposition of the laser energy that leads to the acoustic pressure pulse. Several laser systems and CO_2-TEA lasers in particular often emit sharp pulses that are followed by a relatively long tail, which frequently contains a significant portion of the total energy that is delivered. This distribution of energy has minimal effects on the peak temperatures that are reached by the sample because the time in the initial pulse and in the tail are short compared to thermal conduction times. However, Sigrist and Kneubühl stated that they felt that only the energy in the initial peak and not the energy in the long tail of the laser pulse would con-

tribute to the thermoelastic effect. Extension of the model to include temporal effects in the deposition of laser energy is warranted. Such effects may underly the variability that has been noted in damage thresholds (cf. Table I).

Cleary and Hamrick (Ref. 11) also published work dealing with laser-induced acoustic transients in the mammalian eye. They felt that thermal mechanisms did not adequately explain damage in retina resulting from Q-switched ruby laser pulses. They developed an acoustic model similar to that of Sigrist and Kneubühl (Ref. 9) and compared its predictions with experimental pressure measurements. The pressures were generated by a Q-switched ruby laser impinging on a dye solution having an absorption coefficient of 1000 cm^{-1}. The results should therefore be comparable to those of a CO_2-TEA laser interacting with an aqueous media (absorption coefficient 950 cm^{-1}). Pressures in excess of 100 atm. were measured for a 1 J/cm^2 - 50 ns pulse in this system--a result that was in reasonable agreement with their calculations.

b. Thermal Damage Models

Egbert and Maher summarized early work on epithelial damage thresholds and discussed the empirical modified critical temperature and damage integral models that have been used to correlate threshold damage (Ref. 4). Both of these models depend on being able to determine the temperature history at some position in the epithelium, which is accomplished by solving the heat conduction equation in a straightforward manner. Reed extended Egbert and Maher's results for the critical temperature model and gave an empirical fit to the data they analyzed in terms of the exposure duration and absorption coefficient (Ref. 3). Indeed, it was on the basis of Reed's empirical model that Zuclich et al concluded that damage from short-pulse CO_2 laser radiation was purely thermal. While Reed's model apparently can correlate damage from very short pulses, the damage inte-

gral model has led to inconclusive predictions in this regime (Ref. 7). A major difficulty with all of these models is that, since they consist of empirical fits to experimental data, they provide little or no physical insight into the damage mechanism and there is no *a priori* means to judge their range of applicability.

We have considered a new thermal damage model in which damage is associated with the occurrence of an endothermic phase transition (Refs. 5,6). It provides an excellent correlation of damage data for exposures ranging between 1 ms and 10 s. Moreover, it provides a physical explanation of the weak dependence of critical temperature on exposure duration evidenced in Egbert and Maher's empirical equation (Ref. 4). In particular, this dependence is due to heat conduction that takes place during the phase transition (Refs. 5,6). The model is described in detail in the above noted references. Briefly, a beam having uniform irradiance is incident on and absorbed at the surface of a semi-infinite slab. This causes the temperature to increase according to the well-known equations (Ref. 12). When the surface temperature reaches the transition temperature, a phase transition takes place at the front surface. The surface temperature remains constant during the time that the transition occurs. The amount of energy per unit surface area that goes into the transition is the difference between the incident flux and the rate at which heat is conducted into the cornea, integrated over the time during which the transition occurs. This particular model can be solved analytically, and the results suggest that endothermic phase transitions can explain observed damage thresholds at least between 1 ms and 10 s. The model suggests that the phase transition occurs at a temperature rise of ~33 °C, a figure not far from the calculated temperature rise for the single 80 ns exposure (cf. Table I). Thus, the thermal damage cannot be ruled out for the short exposures. The model should be extended to more

realistic conditions by accounting for the radial heat flow associated with a Gaussian irradiance profile. The model should also be extended to include multiple pulse exposures.

5.0 SUMMARY

This report presents single- and multiple-pulse corneal damage threshold data for a high-power CO_2 TEA laser operated in the TEM_{00} mode. The preliminary multiple-pulse data need to be refined in future research due to a slight inconsistency. At near-threshold energy densities, material was photographed being ejected from the anterior corneal surface. The ejection takes place several microseconds after the end of the 80 nanosecond laser pulse. The mechanism of this process is an intriguing mystery to be solved. The histology of near-threshold lesions was presented; it supports aspects of both acoustic and thermal damage mechanisms. Further research should be performed to clarify and combine the findings into a comprehensive model that can explain both thermal and mechanical features of the laser/tissue interaction. As a starting point, it was suggested that the acoustic model of Sigrist and Kneubühl (Ref. 9) be extended to include the temporal effects of the laser energy deposition. In addition, our thermal model which already includes an endothermic phase transition should be broadened to include radial heat conduction and multiple pulse exposures.

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Abstracts (continued)

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